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OXIDATIVE DAMAGE OF SULPHUR DIOXIDE INHALATION IN BRAIN AND LUNGS OF RATS UNDER NATURAL AND LABORATORY CONDITIONS

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ABSTRACT

Effect of sulphur dioxide (SO_2) on antioxidant enzymes and histopathological changes was investigated in brain and lungs of rats of both the sexes under natural and experimental conditions. Naturally exposed, house rats, *Rattus rattus* were collected from agricultural fields alongside the road at Ludhiana, India. Laboratory house rats were exposed to different concentrations of SO_2 (5ppm, 10ppm and 15 ppm for 5 hours /day for 28 days in 1 m^3 exposure chamber). Control rats were exposed to filtered air in a similar chamber. The results showed that SO_2 exposure at all the tested concentrations caused a significant increase in the lipid peroxidation process in the brains and lungs of rats of both the sexes that is supplemented by changes of antioxidant status in these organs as compared to field rats. The results lead to conclusion that SO_2 can cause oxidative damage to brain and lungs of rats. So, SO_2 is a toxin to brain and lungs of mammals as rats have physiology similar to that of humans. Thus, studying the toxic effect of increased concentrations of SO_2 is the need of hour as increasing pollution is a cause of concern for humans as SO_2 is one of the major pollutants in the air.

Keywords: Brain, Lungs, Oxidative damage, Rats, Sulphur dioxide (SO_2)

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Sulphur dioxide (SO_2) is a colourless, toxic, highly reactive, most commonly found air pollutant formed by the combustion of fossil fuels, industrial processes and motor vehicle operations. It produces negative impact on human health (Shen, 2020). It is present in high concentrations in urban and industrial settings and high concentrations of SO_2 could affect functioning of vital organs as was reported by earlier workers (Rall, 1974). SO_2 gas above 3 ppm concentration has a strong, nauseating odour and most of the developed countries have established SO_2 at 2 ppm concentration as the industrial maximal limit for 8 hours per day.

When humans were experimentally exposed to SO_2 in high concentrations it produced symptoms like fatigue, thickened mucous layer of respiratory tract, labored breathing, stomach aches and changes in chemoreceptors and tactile receptors (Meng, 2003). Very little knowledge is available on the toxicity of SO_2 or its derivatives, (Shi and Mao, 1994), but SO_2 may cause oxidative stress to tissues and organs formed by free radical production during oxidation process (Shi, 1994; Meng and Zhang, 1999; Meng, 2003). Several studies were conducted by scientists to show the toxicity of SO_2 at 10 ppm concentration revealed the increased lipid peroxidation in lungs and RBC's of rats (Etlik *et al*., 1995; Gumuslu *et al*., 1998; Yargicogvlu *et al*., 1999).

National Advisory Committee for Acute Exposure Guideline Levels (AEGL), USA for Hazardous Substances under the Federal Advisory Committee Act has given acute exposure guideline level for SO_2 as 3 ppm for 10 hours and 5 ppm for 5 hours. On the basis of this exposure limit three different concentrations of SO_2 were selected for the present study. 5 ppm and 10 ppm concentration of SO_2 represents 10 and 20 fold greater than the typical urban concentration (0.5ppm) and is known to induce harmful effects to respiratory system of healthy individuals. Third concentration of 15 ppm which is beyond the natural exposure and is used to examine the effects of this higher concentration of SO_2 on the health of individuals.In this study, the brain and lungs of rats were examined for oxidative damage and stress and histopathological damage instigated by SO_2 inhalation at three concentrations (5, 10 and 15 ppm) and under field conditions.

MATERIALS AND METHODS

Mature male and female rats of weight 100-150 g were taken. Naturally exposed (Group I), house rat, *Rattus rattus* were procured from agricultural fields alongside the road in Ludhiana, India and acclimatized for 1 month in the laboratory. Laboratory rats were divided into four groups. Each group was further subdivided into two subgroups having 6 male and 6 female rats. Group II (control rats) was exposed to filtered air for 5 hours /day for 28 days in 1 m^3 exposure chamber, Group III, Group IV and Group V were exposed to5ppm, 10 ppm and 15 ppm of SO_2respectively. SO_2 gas was provided to treated rats through a tube located at the top of each chamber and was distributed with the help of a fan in each chamber (Meng and Zhang, 2003). Rats were kept in cages under standard conditions of

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humidity and temperature with light-dark cycle. Food (loose mixture of cracked wheat grains, powdered sugar and edible vegetable oil in ratio 96:2:2) and water were provided to control and treated rats *ad libitum* (Meng and Zhang, 2003). The weight of individual rat was recorded weekly. After 28 days of exposure and deprivation of food for 18 hours rats were dissected. The experiment was performed after the approval of Institutional Animal Ethical Committee, Guru Angad Dev Veterinary and Animal Sciences University Ludhiana, India under protocol No. (GADVASU/2022/IAEC/64/17) dated March 11, 2022.

Measurements of antioxidant enzymes

After dissection, the brain and lungs of male and female rats were removed and weighed. The tissues were sheared in 0.9% saline (chilled) and homogenization was done in 0.1 M phosphate buffer (pH 7.4). The homogenates were centrifuged for 30 min at 1000 rpm at 4^0C to obtain supernatants. The tissue supernatants were used for the assay of different antioxidant enzymes like Superoxide Dismutase (SOD), Glutathione S-transferase (GST), Catalase (CAT), (Glutathione) GSH, Glutathione peroxide (GPx), Glutathione reductase (GR) and Lipid peroxidation (LPO).

SOD activity

Brain and lungs supernatant was used for estimating the activity of SOD by standard method of Marklund and Marklund (1974).One Unit of SOD is the enzyme that causes 50% inhibition of pyrogallol. The activity was calculated as units/mg of tissue protein.

Glutathione-S-transferase (GST) activity

GST activity in tissue homogenate was estimated by the method of Habig *et al.* (1974). The activity was expressed as µmoles of GSH-CDNB conjugate formed/ min/mg protein.

GSH-Px activity

The GSH-Px activityin tissue homogenate was determined by the method of Hafeman *et al.* (1984). One unit of GSH-Px is the enzyme that catalyses the oxidation of reduced glutathione to oxidised glutathione per minute by H_2O_2.

Glutathione reductase (GR) activity

GR enzyme activity was estimated by the method given by Carlberg and Mannervik (1985) in tissue homogenate. Glutathione reductase activity per unit is the enzyme that causes the oxidation of one µmol of NADPH/min. The activity was calculated as µmoles of NADPH oxidized/min/mg protein.

CAT activity

Catalase enzyme activity was estimated by the standard method of Aebi (1983). The absorbance was measured at 240 nm using a spectrophotometer.

LPO activity

Lipid peroxidation (LPO) activity was estimated by the method of Stocks and Dormandy (1971). The values were expressed as nmol MDA produced/g tissue/h using a molar extinction coefficient of MDA as $1.56X10^5$.

GSH activity

GSH activity was estimated by standard method of Jollow *et al*. (1974). Data was expressed as nM/mg of tissue protein.

Protein assay

In the estimations of all the antioxidant enzymes the total soluble protein content was estimated by Lowry *et al*. (1991) taking BSA as standard.

Histological studies

Histopathology of brain and lungs was done according to the standard method of Luna (1968). Brain and lungs of rats were cleared from adhering tissues and fixed in 10% neutral formalin for 24 hours. Then the tissues were dehydrated in ascending grades of ethanol, cleared in xylene and embedding was done in paraffin wax (melting point-58-60°C) for the preparation of blocks. The 5-7µm thick sections were cut serially with the help of microtome and were floated in tissue floatation bath at 40^oC and placed on glass slides rehydrated in descending grades of ethanol, cleared in xylene and stained in haematoxylin-eosin and mounted in DPX. Then slides were studied under light microscope.

Statistical analysis

Statistical Analysis Software (SPSS) was used to analyse the data. The data was subjected to One way Analysis of Variance (ANOVA) with post host Tukey's t-test for making comparisons between naturally exposed and treated group of rats. A value of $P< 0.05$ indicated significant differences.

RESULTS AND DISCUSSION

There were two important considerations for the planning of this experiment. Firstly, rats were exposed to SO_2 for a regular period (5 h/day for 7 days with 19 hours between exposures) with relief periods in between the exposure. Secondly rats are nose breathers and most of inhaled gas is trapped in nasal chambers and only some amount is reaching to the lungs that's why

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higher concentration (15 ppm) of SO_2 was used and their comparison with rats collected from agricultural fields alongside the roads. Both the considerations may provide a repercussion to persons exposed to the gas in an occupational or industrial setting.

The rationale and relevance of exposure of rats to sulphur dioxide is that rat is best mammalian experimental model having physiology similar to human beings and the toxic effect on rats is almost similar as that on humans. Field rats were collected from agricultural fields alongside the road where the emission of sulphur dioxide gas is more which can be compared to humans in direct exposure of vehicular emissions or people working in industrial settings. The field rats have an option to evade by burrowing away but they have to come out of burrow for day today activities like food, water and breeding purpose etc. and then get exposed to vehicular emissions.

Mean body weight, organ weight did not vary significantly between rats of natural group and treated group (5, 10 and 15 ppm). A non-significant decrease in body weight was observed in male and female treated rats as compared to field rats and control rats. No significant difference was observed in the weights of brain and lungs of rats of all the treated groups when compared with field and control rats.

Brain

Tables 1 and 2 represents level of LPO and antioxidant activities of CAT, SOD, GST, GPx, GSH and GR in the brain of male and female rats exposed to SO_2 at three concentrations (5, 10 and 15 ppm) and their corresponding control and naturally exposed (field) rats. At 5 and 10 ppm concentration non-significant decrease in SOD, CAT, GST, GR and GSH activity. While SO_2 at 15 ppm concentration showed significant decrease in SOD, CAT, GPx and GSH activity and non-significant decrease in GST and GR activity. A significant increase in Lipid peroxidation (LPO) activity was observed at 15 ppm concentration in brain of rats of both the sexes as compared to control rats. Higher SO_2 concentration decreased the activities of SOD, CAT, GST, GPx, GSH and increased lipid peroxidation indicating the increased oxidative damage and stress in cells and tissues in present case. These findings demonstrate that SOD and GPx activities were altered by SO_2 in the brains of rats in a dose-dependent manner. At low concentrations, these antioxidant enzymes were significantly unaffected by SO_2, whereas at higher concentrations, SO_2 caused a significant decrease in their activities.The fact that the brain is an aerobic tissue makes it susceptible to oxidative stress brought on by oxygen free radicals. Despite being an organ with one of the greatest oxygen consumption rates in the body, using around one-fifth of the body's total oxygen need, it is not particularly enriched in any antioxidant enzymes when compared to other organs. Because SOD can catalyse the breakdown of superoxide radicals to form hydrogen peroxide, the brain may be more susceptible to increased free radical damage as a result of the antioxidant enzymes' decreased activity. According to van Loon *et al.* (1986), SOD is thought of as the first line of defence against oxygen poisoning. According to Chance *et al.* (2020), se-dependent GPx and CAT are the main hydrogen peroxide scavengers. Numerous studies have reported the toxic effect of SO_2 on nervous system and respiratory system of mammals and lungs and brain tissues as sites for free radical generation and damage (Meng, 2003; Meng *et al*., 2002a; Meng *et al.,* 200b).Bakurt (1994) also reported decreased SOD level in the brain, lungs and erythrocytes of rats after SO_2 exposure.

Lungs

Tables 3 and 4 represent antioxidant activities of SOD, CAT, GST, GPx, GSH and GR in the lungs of male and female rats exposed to SO_2 at three concentrations (5, 10 and 15 ppm) and their corresponding control and naturally exposed (field) rats. 5 and 10 ppm concentration showed significant decrease in SOD, CAT, GSH and GR activity non-significant decrease in GST and GPx activity. While SO_2 at 15 ppm showed significantly decreased SOD, GPx and GSH activity and non-significant reduction in GST and GR activity and a significant increase in LPO activity in lungs from male and female rats as compared to control rats. Decreased level of SOD will decrease the production of hydrogen peroxide and it also reduces the protection against free radicals. The respiratory system is thought to be toxic to $SO_2$and its in vivo derivatives*/bisulfite/sulfite, which can also produce allergic reactions, the most frequent of which is bronchoconstriction in asthmatics (Ceballos-Picot *et al.,* 1992). In addition to acting as a co-mutagen and carcinogen to link to lung cancer, it may cause cytogenetic damage in human lymphocytes (Lester, 1995; Meng and Zhang, 1990,1992,1999). The most frequent clinical manifestations linked to SO_2 exposure include bronchospasm, pulmonary edoema, pneumonitis, and acute airway blockage and damage to nervous system (Chen *et al*., 2007).

The findings of Medeiros (1983) also showed the reduction in antioxidation status and increased lipid peroxidation after SO_2 exposure in mice are also in agreement with our results. Reduction in CAT and GPx level will lessen the security of cells against the free radicals. Glutathione peroxidase is known for the reduction of hydro peroxides to H_2O through oxidation of reduced GSH into Glutathione disulphide (GSSH). The decrease in the level of GST, GSH and GPx at higher concentration of SO_2 was found to be dose

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Table 1. Effect of SO_2 on antioxidant parameters in brain of male rats

Group	Treatment	Antioxidant Parameters						
		Superoxide dismutase (SOD)	Catalase (CAT)	Glutathione-S-transferase (GST)	Glutathione Peroxidase (GPx)	Glutathione (GSH)	Glutathione reductase (GR)	Lipid peroxidation (LPO)
I	Naturally exposed rats	7.83 ±0.27^{c}	6.65± 0.12^{c}	0.46± 0.004^{a}	0.56± 0.01^{b}	45.87 ± 0.46^{b}	0.04±0.003^{b}	0.66± 0.02^{a}
II	Control	8.65 ±0.08^{c}	8.95 ± 0.08^{d}	0.48 ± 0.007^{b}	0.87 ± 0.005^{b}	48.65 ± 0.16^{c}	0.05±0.0003^{c}	0.37 ± 0.01^{a}
III	5ppm SO_2	7.37 ±0.20^{c}	6.61 ±0.18^{c}	0.45 ±0.005^{a}	0.69± 0.01^{a}	45.74 ±0.18^{b}	0.04±0.003^{ab}	0.48 ±0.01^{a}
IV	10ppm SO_2	6.28 ±0.21^{b}	5.69 ±0.14^{b}	0.41±0.003^{a}	0.54±0.007^{a}	41.60±0.39^{a}	0.04±0.0006^{ab}	0.51±0.09^{a}
V	15ppm SO_2	3.79 ±0.10^{a}	3.91 ±0.27^{a}	0.42±0.004^{a}	0.34 ±0.025^{ab}	40.42 ± 0.15^{a}	0.03±0.001^{a}	0.97 ±0.09^{b}

Values are shown as mean±SE of 6 animals in each group
^{abcd} represents significant difference between treatments at p≤0.05 as compared to control and naturally exposed rat.
SOD, CAT and GPx expressed as (U/mg protein), GST (µmoles of GSH/mg protein), GR (µmoles of NADPH oxidized/min/mg protein), Lipid peroxidation (nmol MDA/100 mg tissue)

Table 2. Effect of SO_2 on antioxidant parameters in brain of female rats

Group	Treatment	Antioxidant Parameters						
		Superoxide dismutase (SOD)	Catalase (CAT)	Glutathione-S-transferase (GST)	Glutathione Peroxidase (GPx)	Glutathione (GSH)	Glutathione reductase (GR)	Lipid peroxidation (LPO)
I	Naturally Exposed rats	9.34±0.31^{b}	8.60±0.16^{c}	0.45±0.004^{a}	0.72 ± 0.03^{a}	48.35± 0.14^{c}	0.05± 0.004^{c}	0.34 ±0.003^{a}
II	Control	9.38±0.30^{b}	8.71±0.13^{c}	0.56±0.06^{b}	0.79 ± 0.007^{b}	48.63±0.11^{c}	0.06 ± 0.002^{c}	0.377±0.009^{a}
III	5 ppm SO_2	5.74±0.14^{a}	5.42±0.14^{b}	0.48±0.14^{a}	0.77 ± 0.01^{b}	41.5 ±0.42^{ab}	0.042±0.0008^{a}	0.76 ± 0.07^{b}
IV	10 ppm SO_2	6.076±0.11^{a}	4.9±0.22^{ab}	0.46±0.01^{a}	0.72 ± 0.016^{a}	42.25 ± 0.47^{b}	0.043±0.0006^{a}	0.80±0.032^{bc}
V	15 ppm SO_2	3.49±0.44^{c}	4.61±0.16^{a}	0.43±0.003^{b}	0.45± 0.04^{a}	40.39 ± 0.28^{a}	0.05±0.002^{b}	0.90 ± 0.04^{c}

Values are expressed as mean±SE of 6 animals in each group
^{abcd} represents significant difference between treatments at p≤0.05 as compared to control and naturally exposed rat.
SOD, CAT and GPx expressed as (U/mg protein), GST (µmoles of GSH /mg protein), GR (µmoles of NADPH oxidized/min/mg protein), Lipid peroxidation (nmol MDA/100 mg tissue)

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Table 3. Effect of SO_2 on antioxidant parameters in lungs of male rats

Group	Treatment	Antioxidant Parameters						
		Superoxide dismutase (SOD)	Catalase (CAT)	Glutathione-S-transferase (GST)	Glutathione Peroxidase (GPx)	Glutathione (GSH)	Glutathione reductase (GR)	Lipid peroxidation (LPO)
I	Naturally Exposed rats	12.73±0.21^{c}	9.41±0.01^{c}	0.48±0.004^{b}	0.74±0.016^{a}	46.34± 0.05^{c}	0.067±0.001^{b}	0.47± 0.03^{a}
II	Control	12.98±0.21^{c}	9.87±0.02^{c}	0.55±0.02^{b}	0.77±0.006^{a}	47.77±0.07^{c}	0.064±0.001^{b}	0.46±0.048^{a}
III	5 ppm SO_2	5.75±0.07^{b}	4.85±0.09^{b}	0.46±0.01^{a}	0.71±0.01^{a}	41.81±0.28^{ab}	0.05±0.001^{a}	0.87±0.072^{c}
IV	10 ppm SO_2	5.35±0.17^{ab}	4.02±0.23^{a}	0.45±0.01^{a}	0.70±0.008^{a}	42.60±0.39^{b}	0.054±0.001^{a}	0.60±0.003^{b}
V	15 ppm SO_2	4.21±0.33^{a}	4.39± 0.21^{ab}	0.47±0.008^{a}	0.60± 0.06^{a}	41.16±0.21^{a}	0.05±0.0009^{a}	0.88±0.03^{c}

Values are expressed as mean±SE of 6 animals in each group
^{abcd} represents significant difference between treatments at p≤0.05 as compared to control and naturally exposed rat.
SOD, CAT and GPx expressed as (U/mg protein), GST (µmoles of GSH /mg protein), GR (µmoles of NADPH oxidized/min/mg protein), Lipid peroxidation (nmol MDA/100 mg tissue)

Table 4. Effect of SO_2 on antioxidant parameters in lungs of female rats

Group	Treatment	Antioxidant Parameters						
		Superoxide dismutase (SOD)	Catalase (CAT)	Glutathione-S-transferase (GST)	Glutathione Peroxidase (GPx)	Glutathione (GSH)	Glutathione reductase (GR)	Lipid peroxidation (LPO)
I	Naturally Exposed rats	12.67 ± 0.19^{c}	9.48 ± 0.12^{b}	0.56±0.001^{c}	0.84 ± 0.01^{b}	47.77±0.14^{d}	0.05±0.009^{b}	0.46 ±0.001^{a}
II	Control	12.74 ± 0.17^{c}	9.74 ± 0.13^{b}	0.57±0.003^{c}	0.83±0.017^{b}	48.22±0.12^{d}	0.05±0.0009^{b}	0.47±0.011^{a}
III	5 ppm SO_2	5.98 ± 0.24^{b}	4.57 ± 0.15^{a}	0.44±0.016^{b}	0.72 ±0.011^{a}	41.42 ±0.30^{c}	0.041±0.001^{a}	0.92 ± 0.02^{c}
IV	10 ppm SO_2	6.07 ± 0.11^{b}	4.05 ± 0.23^{a}	0.42 ±0.014^{a}	0.75 ±0.015^{a}	42.23 ±0.24^{c}	0.042±0.001^{a}	0.60±0.006^{b}
V	15 ppm SO_2	4.95 ± 0.21^{a}	3.57 ± 0.38^{a}	0.48±0.001^{b}	0.70 ± 0.01^{a}	40.28 ±0.25^{a}	0.043±0.0009^{a}	0.92 ± 0.02^{c}

Values are expressed as mean±SE of 6 animals in each group
^{abcd} represents significant difference between treatments at p≤0.05 as compared to control and naturally exposed rat.
SOD, CAT and GPx expressed as (U/mg protein), GST (µmoles of GSH /mg protein), GR (µmoles of NADPH oxidized/min/mg protein), Lipid peroxidation (nmol MDA/100 mg tissue)

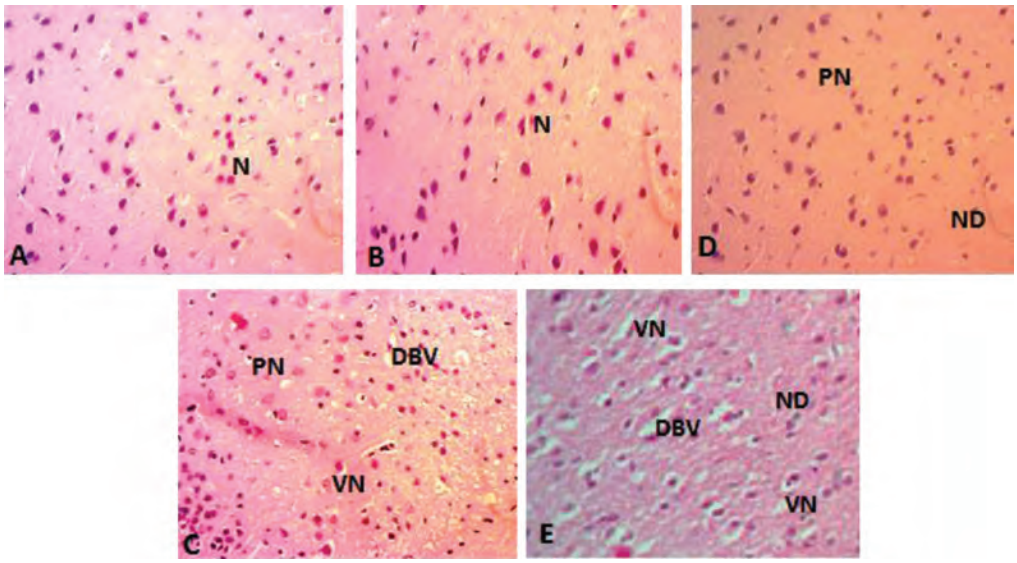


Fig. 1. Brain Tissue Section from Control rat and naturally exposed rats showed normal nerve or glial cells(A,B) in 5 ppm SO_2 exposed group brain showed pyknotic neurons (PN) and vacuolated neurocytes (VN) (Fig 1 C) 10 ppm and 15 ppm SO_2 exposed group showed gliosis(G), degenerated neurons, dilated blood vessels (DBV) and degenerated neurons (Fig. 1 D,E) observed by light microscopy with X400 magnification

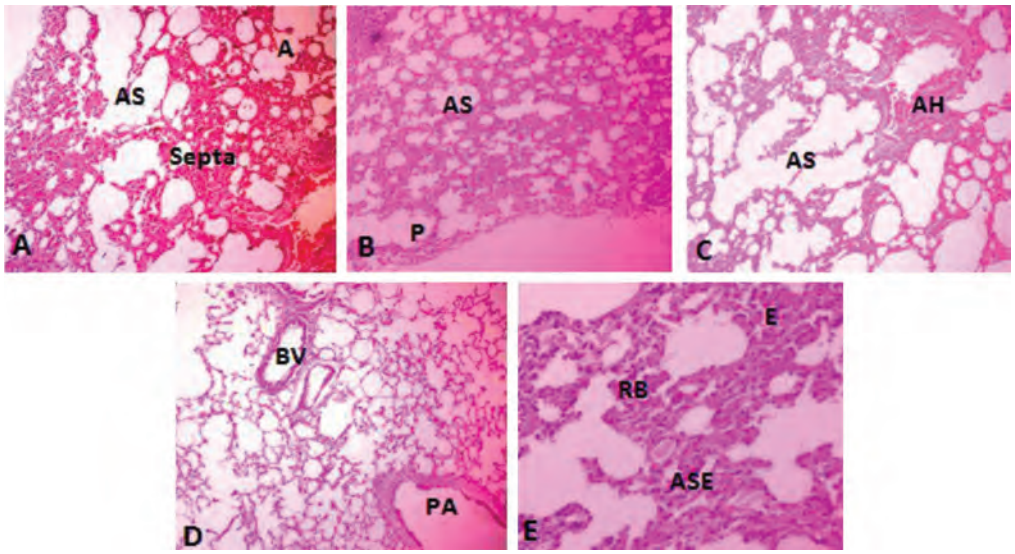


Fig. 2. Lung Section from Control and naturally exposed rats showing normal alveoli, alveolar Sacs (AS), pneumocytes (P), normal alveolar septa and blood vessels (BV), no inflammation (A,B) 5 ppm SO_2 exposed group showed increased alveolar spaces, eedema and thickened wall of alveoli (TW), alveolar wall destruction(D) (Fig. 2 C) 10 ppm SO_2 exposed group showed alveolar space exudation (ASE), haemorrhage in alveoli and blood vessels in the alveolar space (Fig. 2 D) In 15 ppm SO_2 exposed group showing alveolar space exudation (ASE), alveolar hemorrhage (AH), respiratory bronchiole (RB), thickened interalveolar septa and eedema (E) in the alveoli (Fig. 2 E) observed by light microscopy with X400 magnification

dependent. Leung *et al*. (1985) and Lovati *et al*. (1996) also reported similar decrease in levels of GST, GSH and GPx in their studies on effect of SO_2 inhalation on rats. Decreased level of GSH after SO_2 inhalation was also shown by some other studies on rats (Lovati *et al*., 1996).Under normal conditions, cells cope with free radicals with the help of antioxidant enzymes like SOD, CAT, GSH, GPx (Ceballos-Picot *et al*., 1992; Singh and Pathak, 1990). Studies conducted by Haider *et al*. (1981) and Etlik *et al*. (1995) showed increased lipid peroxidation in brain of rats and erythrocytes of guinea pigs exposed to SO_2. Tyler (1975) also accounted for increased lipid peroxidation in mitochondria after exposure to derivatives of SO_2 in laboratory conditions.

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Increased lipid peroxidation seems to be indicator of several disorders in cells, tissues or organs and it might be involved in different diseased state of cell such as aging, nervous disorders etc. (Meng *et al*., 2002a; Meng *et al*., 2002b). Increased lipid peroxidation may have harmful effects on composition and function of biological membranes (Gupta *et al*., 1991). A number of studies have been reported to show the increased lipid peroxidation in brain of rats and RBCs of guinea pigs (Etlik *et al*. ,1995; Haider *et al*., 1981; Gumuslu *et al*., 1998; Yargicogvlu *et al*., 1999). SO_2 toxicity may involve oxidative stress to cells and tissues because of the production of free radicals during oxidation process (Shapiro, 1977; Shi and Mao, 1994).

Roemer and Van Wijnen's (2021) conducted an epidemiological study to assess the short-term impacts of air pollutants (NO_2 and SO_2) both close to roadways and farther away. A sample of 4352 Amsterdam inhabitants (n = 4352) who lived "along roads with more than 10,000 motorized vehicles per day" and was compared with normal population provided the data for this study.They found higher levels of SO_2 and NO_2 and carbon monoxide at the traffic-influenced measurement sites compared with the normal sites. Other studies have also investigated the effects of multiple pollutants near roads on outcomes for respiratory health and allergic disease (Gauderman, 2005; Morgenstern, 2008; Rosenlund, 2009a; McClellan *et al*., 2012; Gruzieva, 2013; Willers, 2013).

Histologically, brain tissue section of control rats and naturally exposed rats showed normal nerve or glial cells. In 5 ppm SO_2 exposed group, brain showed pyknotic neurons and vacuolated neurocytes, In 10 ppm and 15 ppm SO_2 exposed group brain showed gliosis, degenerated neurons, dilated blood vessels and degenerated neurons (Fig. 1). Our findings are in agreement with that of Liu *et al*. (2022).

Histologically, lung section of control and naturally exposed rats showing normal alveolar sacs (AS), normal pneumocytes (P), normal alveolar septa and blood vessels, no inflammation and no alveolar space exudation. While in 5 ppm SO_2 exposed group alteration were seen like increased alveolar spaces, oedema and thickened wall of alveoli and in 10 ppm SO_2 exposed group showing alveolar space exudation (ASE), haemorrhage in alveoli and blood vessels in the alveolar space. 15 ppm SO_2 exposed group showed alveolar space exudation (ASE), alveolar haemorrhage (AH), thickened inter-alveolar septa and oedema (E) in the alveoli (Fig. 2). Haider *et al*. (1981) also reported similar findings.

It can be concluded from present study that SO_2 exposure caused a significant increase in the lipid peroxidation process in the brains and lungs of rats of both sexes that is supplemented by reduction in antioxidant status in a dose dependent manner in laboratory rats when compared with field rats. Secondly, the effects of SO_2 on mammals are multifaceted and it is a toxin to brain and lungs besides other organs. Further work needs to be done to understand the toxic role of SO_2 on all organs of mammals. And to study the toxic effect of increased concentrations of SO_2 is the need of hour as increasing pollution is a course of concern for humans as SO_2 is one of the major pollutants in the air.

Authors' contribution

Conceptualization and designing of the research work (PS, NS); Execution of field/lab experiments and data collection (PS); Analysis of data and interpretation (PS,NS); Preparation of manuscript(PS, NS).

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